## **Amendments to the Claims**

Please cancel Claims 1 through 68. Add the following new claims:

- 1. through 68. (Cancelled)
- 69. (New) A method for controlling aberrant cell proliferation comprising
- a) contacting a cell population comprising aberrantly proliferating cells with at least one Chk1 activator for from about 30 minutes to about 96 hours wherein the Chk1 activator is selected from the group consisting of

mechlorethamine, cyclophosphamide, ifosfamide, melphalan, chlorambucil, carmustine (BCNU), lomustine (CCNU), semustine (methyl-CCNU), triethylenemelamine (TEM), triethylene thiophosphoramide (thiotepa), hexamethylmelamine (HMM, altretamine), busulfan, dacarbazine (DTIC), methotrexate, trimetrexate, pemetrexed (multi-targeted antifolate), 5-fluorouracil (5-FU), fluorodeoxyuridine, gemcitabine, cytosine arabinoside (AraC, cytarabine), 5-azacytidine, 2,2'-difluorodeoxycytidine, 6-mercaptopurine, 6-thioguanine, azathioprine, 2'-deoxycoformycin (pentostatin), erythrohydroxynonyladenine (EHNA), a fludarabine salt, 2-chlorodeoxyadenosine (cladribine, 2-CdA), camptothecin (CPT), topotecan, irinotecan, etoposide, teniposide, vinblastine, vincristine, vinorelbine, actinomycin D, doxorubicin, bleomycin, 5-bromodeozyuridine, 5-iododeoxyuridine, bromodeoxycytidine, cisplatin, carboplatin, oxaliplatin, hydroxyurea, and x-ray radiation

in an amount sufficient to substantially synchronize cell cycle arrest among said aberrantly proliferating cells at a target phase, and

b) upon achieving said substantial synchronization of cell cycle arrest among said aberrantly proliferating cells, contacting said cell population with a selective Chk1 inhibitor for from up to about 1 hour to up to about 72 hours wherein the selective Chk1 inhibitor is a compound of formula

$$W$$
 $X^1$ 
 $X^2$ 
 $Z$ 

wherein X1 is null,-O-,-S-,-CH2-, or - N (R1)-;

X2 is -O-, -S-, or-N(R1)-; Y is O or S; or =Y represents two hydrogen atoms attached to a common carbon atom; W is selected from the group consisting of heteroaryl, aryl, heterocycloalkyl, cycloalkyl, and C13 alkyl substituted with a heteroaryl or aryl group;

W and Z are selected from the group consisting of hydro, aryl, and heteroaryl; wherein said aryl groups of W and Z are optionally substituted with one to four substituents represented by R2, said heteroaryl groups of W and Z are optionally substituted with one to four substituents represented by R5, and said heterocycloalkyl and cycloalkyl groups of W are optionally substituted with one to two substituents represented by R6;

R1 is selected from the group consisting of hydro, C1-6alkyl, C2-6alkenyl, C2-6alkynyl, and aryl;

R2 is selected from the group consisting of halo, optionally substituted C1-6alkyl, C2-6alkenyl, OCF3, NO2, CN, NC, N(R3)2, OR3, CO2R3, C(0) N (R3)2, C (O)R3, N (R1) COR3, N (R1) C (O) OR3, N (R3) C (O) OR3, N (R3) C (O) C1-3alkyleneC(O)R3, N (R3) C (O) C1-3alkyleneC(O)C1-3alkyleneOR3, N(R3)C(O)C1-3alkyleneNHC(O)-OR3, N(R3)C(O)C1-3alkyleneSO2NR3, C1-3alkyleneOR3, and SR3;

R3 is selected from the group consisting of hydro, C1-6alkyl, C2-6alkenyl, cycloalkyl, aryl, heteroaryl, SO2R4, C1-6alkyl substituted with one or more of halo, hydroxy, aryl, heteroaryl, heterocycloalkyl, N (R4) 2, and SO2R4, C1-3alkylenearyl, C1-3alkyleneheteroaryl, C1-3alkyleneC3-8heterocycloalkyl, C1-3alkyleneSO2aryl, optionally substituted Cl-3alkyleneN(R4)2, OCF3, C1-3alkyleneN(R4)3+, C3-8heterocycloalkyl, and CH(C1 3alkyleneN(R4)2)2, or two R3 groups are taken together to form an optionally substituted 3-to 6-membered aliphatic ring;

R4 is selected from the group consisting of hydro, C1-6alkyl, cycloalkyl, aryl, heteroaryl, C1-3-alkylenearyl, and SO2C1-6alkyl, or two R4 groups are taken together to form an optionally substituted 3-to 6-membered ring;

R5 is selected from the group consisting of Cl-6alkyl, aryl, N(R3) 2, OR3, halo, N3, CN, C1-3alkylenearyl, C1-3alkyleneN(R3) 2, C(O)R3, and

R6 is selected from the group consisting of halo and C1-6alkyl; or a pharmaceutically acceptable salt thereof

in an amount sufficient to substantially abrogate said cell cycle arrest.

- 70. (New) The method of claim 69, wherein said cell population is contacted with a Chk1 activator for from about 30 minutes to about 48 hours.
- 71. (New) The method of claim 70, wherein said cell population is in a human.
- 72. (New) The method of Claim 69, wherein said aberrantly proliferating cells are cancerous.
- 73. (New) The method of claim 72, wherein said cancerous cells comprise cells from non-small cell lung cancers or colon cancers.